

A rare clinical presentation of Epstein-Barr virus

ABSTRACT

In this letter we report a case of Guillain-Barré syndrome associated with infectious mononucleosis. A 20-year-old woman presented to our hospital with weakness of legs and arms. She had felt weakness in her legs for three days and in arms for one day. She had one week history of pharyngitis, muscle pain, and fever. Serological data confirmed infectious mononucleosis. Electrophysiological findings and cerebrospinal fluid examinations confirmed Guillain-Barré syndrome. She was treated with immunoglobulin, methylprednisolone and acyclovir. Paresis improved five days after the end of immunoglobulin therapy. Epstein-Barr virus infection and infectious mononucleosis must be considered among patients with Guillain-Barré syndrome.

Keywords: Guillain-Barré syndrome, infectious mononucleosis.

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TO THE EDITOR

A 20-year old woman admitted to our hospital reporting weakness, which had begun three days ago at her legs. She also reported weakness at her arms, which started one day ago. She had one week history of pharyngitis, muscle pain, and fever around 38° C.

Neurological examination revealed paresis of (Medical Research Council) MRC grade 3-4, with absent deep tendon reflexes. Cerebrospinal fluid revealed increased protein 80 mg/dL (15-45 mg/dL) with 10/mm³ leukocytes and a glucose level of 52 mg/dL (40-70 mg/dL). Electrodiagnostic studies were performed on days 4 and 11 of weakness. Absent F waves and normal motor latencies were observed. Concurrent neurological findings, results of cerebrospinal fluid analysis, and nerve conduction studies were compatible with axonal form of Guillain-Barré syndrome (GBS). Bacterial cultures were negative. Complete blood count, serum electrolytes, and renal function tests were normal. Aspartate transaminase was 96 IU/L (5-35 IU/L) and alanine transaminase 104 IU/L (0-55 IU/L). Urinary excretion of porphyrins was normal. Serologic tests for HIV, VDRL, CMV, HSV, hepatitis A, hepatitis B, hepatitis C, b Borrelia, and Brucella were all negative. VCA-IgM

and VCA IgG were positive. Anti EBNA IgM was negative, Anti EA-D IgG was positive. Also serological data confirmed infectious mononucleosis (IM). She was treated for GBS and IM with immunoglobulin IV 0.4 g/kg/d for 5 days, methylprednisolone IV 1 g for 3 days, and acyclovir IV 750 mg q8h for 2 weeks. Paresis improved 5 days after the end of immunoglobulin therapy, but facial paresis did not recovered.

Epstein-Barr virus (EBV) infects more than 98% of world's adult population, primary EBV infection occurs usually during childhood. EBV enters the body by infecting epithelial cells in the oropharynx. IM is a primary clinical syndrome of EBV, which occurs with fever, pharyngitis, fatigue, lymphadenopathy, splenomegaly, and elevated liver functional tests.¹ Most IM is a self limited disease, but it could cause many systemic complications, such as cardiac, renal, hepatic, hematological, immunological, respiratory, and neurological. Neurological complications account for 2-5% of patients.^{2,3} The most seen neurological complications of IM are encephalitis, meningitis, GBS with the involvement of cranial nerves, cerebellitis, myelitis, and cranial neuropathies. As we know, the clinical picture of GBS is usually a generalized polyradiculoneuropathy affecting limbs proximally and distally and commonly spreading to involve facial and other cranial nerves.^{2,3}

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Although the effect of methylprednisolone and acyclovir treatment in IM and GBS has not been investigated in randomized studies, patients treated with corticosteroids and acyclovir seem to be improved.^{2,4} In our case, paresis improved five days after the end of immunoglobulin therapy, but facial paresis did not recovered.

As a conclusion, the present case indicates that EBV infection that causes IM must be considered among the possible causes in patients with GBS.

REFERENCES

1. Straus SE, Cohen JL, Tosato G, Meier J. NIH conference. Epstein-Barr virus infections: biology, pathogenesis, and management. *Ann Intern Med* 1993; 118(1):45-58.
2. Majid A, Galetta SL, Sweeney CJ *et al.* Epstein-Barr virus myel radiculitis and encephalomyel radiculitis. *Brain* 2002; 125:159-65.
3. Jenson HB. Acute complications of Epstein-Barr virus infectious mononucleosis. *Curr Opin Pediatr* 2000; 12(3):263-8.
4. Finsterer J. Treatment of immune-mediated, dysimmune neuropathies. *Acta Neurol Scand* 2005; 112(2):115-25.